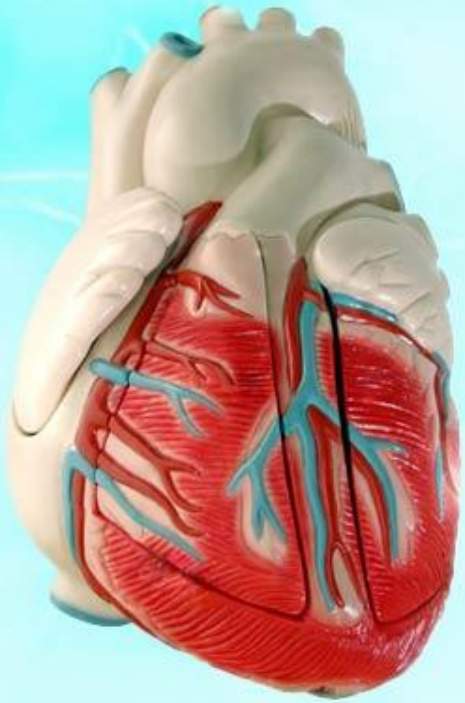

بسم الله الرحمن الرحيم

Cor pulmonale

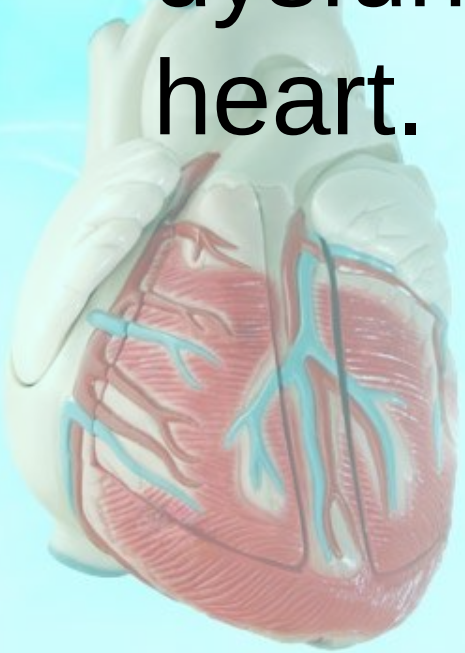
Dr hamid reza javadi



Definition

- Cor pulmonale ;*pulmonary heart disease*; is defined as dilation and hypertrophy of the right ventricle (RV) in response to diseases of the pulmonary vasculature and/or lung parenchyma.

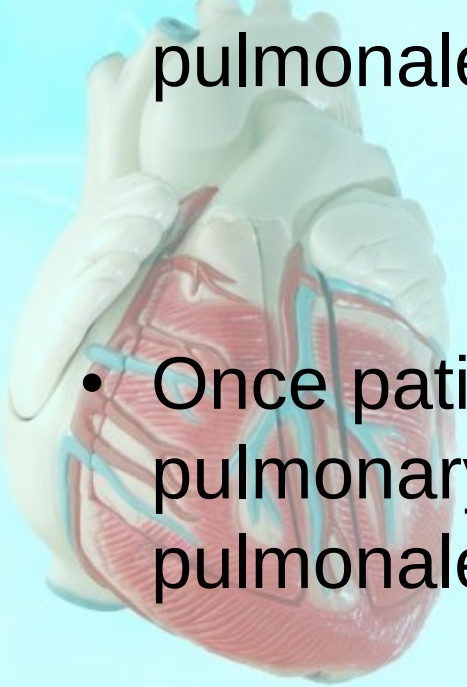
- this definition has excluded congenital heart disease and those diseases in which the right heart fails secondary to dysfunction of the left side of the heart.



Etiology and Epidemiology

- Cor pulmonale develops in response to acute or chronic changes in the pulmonary vasculature and/or the lung parenchyma that are sufficient to cause pulmonary hypertension.
- The true prevalence of cor pulmonale is difficult to ascertain for two reasons:
- 1- not all cases of chronic lung disease will develop cor pulmonale
- 2- our ability to diagnose pulmonary hypertension and cor pulmonale is relatively insensitive.

- chronic obstructive pulmonary disease (COPD) and chronic bronchitis are responsible for approximately 50% percent of the cases of cor pulmonale.
- Any disease that affects the pulmonary vasculature or parenchyma can lead to cor pulmonale.



- Once patients with chronic pulmonary or pulmonary vascular disease develop cor pulmonale, their prognosis worsens.

Table 227-6 Etiology of Chronic Cor Pulmonale
Diseases Leading to Hypoxic Vasoconstriction
Chronic bronchitis
Chronic obstructive pulmonary disease
Cystic fibrosis
Chronic hypoventilation
Obesity
Neuromuscular disease
Chest wall dysfunction
Living at high altitudes

Diseases That Cause Occlusion of the Pulmonary Vascular Bed
Recurrent pulmonary thromboembolism
Primary pulmonary hypertension
Venocclusive disease
Collagen vascular disease
Drug induced lung disease

Diseases That Lead to Parenchymal Disease
Chronic bronchitis
Chronic obstructive pulmonary disease
Bronchiectasis
Cystic fibrosis
Pneumoconiosis
Sarcoid
Idiopathic pulmonary fibrosis

Pathophysiology and Basic Mechanisms

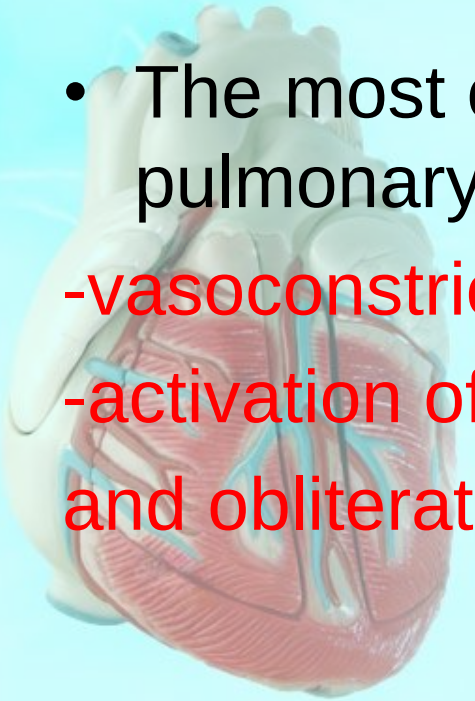
- the common pathophysiologic mechanism is pulmonary hypertension that is sufficient to lead to RV dilation, with or without concomitant RV hypertrophy.

- The most common mechanisms that lead to pulmonary hypertension, including

-vasoconstriction,

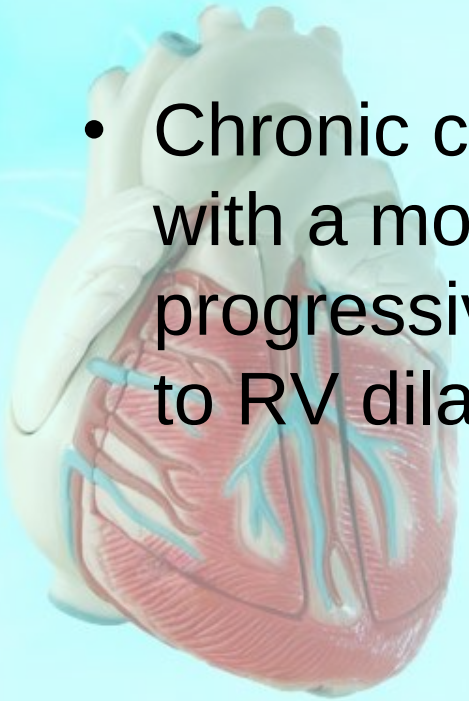
-activation of the clotting cascade,

and obliteration of pulmonary arterial vessels.



- Acute cor pulmonale occurs after a sudden and severe stimulus (e.g., massive pulmonary embolus), with RV dilatation and failure but no RV hypertrophy.

- Chronic cor pulmonale, however, is associated with a more slowly evolving and slowly progressive pulmonary hypertension that leads to RV dilation and hypertrophy.

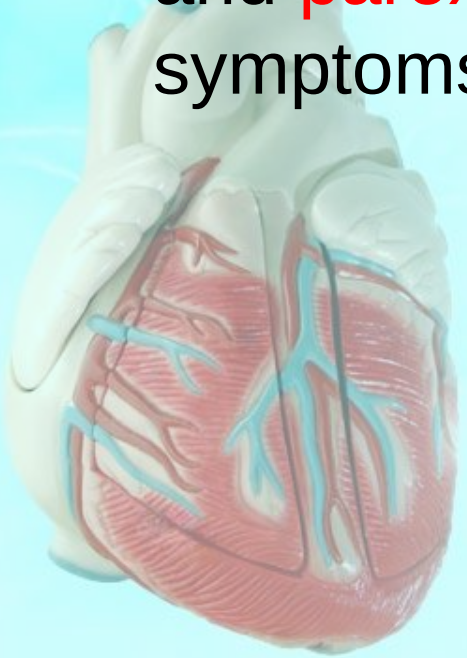


- The severity of the pulmonary artery hypertension and the onset of RV failure are influenced by multiple factors including:

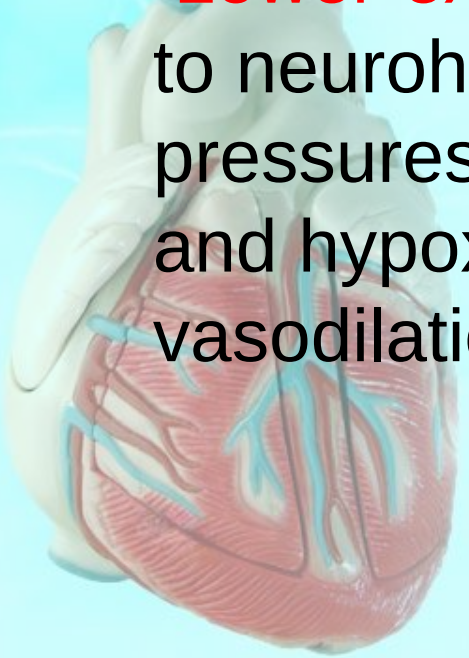
- hypoxia secondary to alterations in gas exchange,
- hypercapnia,
- acidosis,
- as well as alterations in RV volume overload that are affected by exercise, heart rate, polycythemia, or increased salt and retention because of a fall in cardiac output.

Clinical Manifestations

- Symptoms :
- related to the underlying pulmonary disorder.
- Dyspnea, the most common symptom.
- Orthopnea
- and paroxysmal nocturnal dyspnea are rarely symptoms of isolated right HF.



- Tussive or effort-related syncope may occur in patients with cor pulmonale with severe pulmonary hypertension.
- The abdominal pain and ascites that occur with cor pulmonale are similar to the right heart failure that ensues in chronic HF.
- Lower-extremity edema may occur secondary to neurohormonal activation, elevated RV filling pressures, or increased levels of carbon dioxide and hypoxia, which can lead to peripheral vasodilation and edema formation.



- Signs:

-tachypnea,

-elevated jugular venous pressures,

-hepatomegaly,

-and lower-extremity edema.

-Patients may have prominent *v* waves in the jugular venous pulse as a result of tricuspid regurgitation.

-RV heave palpable along the left sternal border or in the epigastrium.

-A systolic pulmonary ejection click may be audible to the left of the upper sternum.

-The increase in intensity of the holosystolic murmur of tricuspid regurgitation with inspiration ("Carvallo's sign") may be lost as RV failure worsens.

-Cyanosis is a late finding in cor pulmonale and is secondary to a low cardiac output with systemic vasoconstriction and ventilation-perfusion mismatches in the lung.

Diagnosis

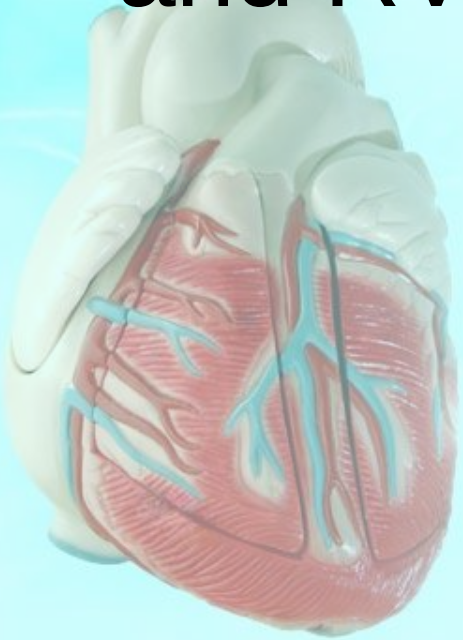
- The most common cause of right heart failure is not pulmonary parenchymal or vascular disease, but left heart failure.
- Therefore it is important to evaluate the patient for LV systolic and diastolic dysfunction.

- The ECG in severe pulmonary hypertension shows:

-P pulmonale,

-right axis deviation,

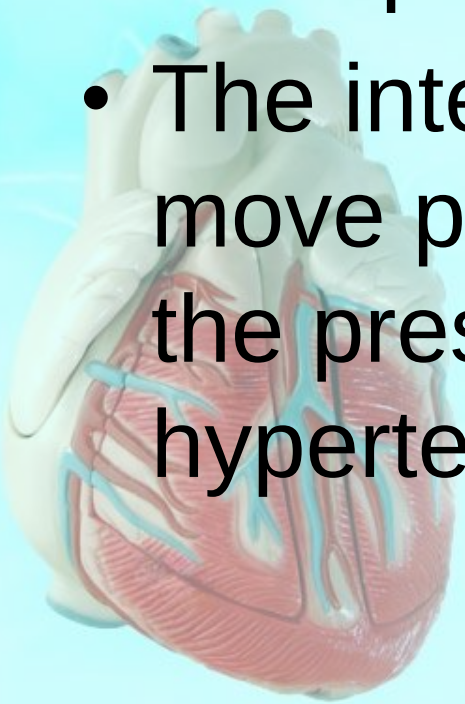
-and RV hypertrophy.



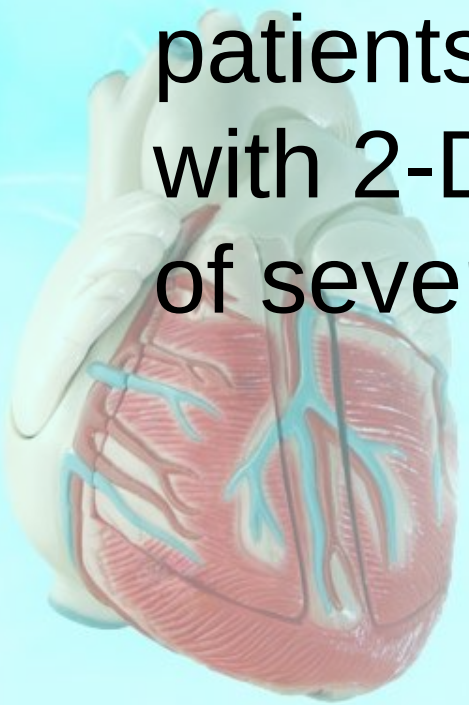
- Radiographic examination of the chest may show enlargement of the main pulmonary artery, hilar vessels, and the descending right pulmonary artery.
- Spiral CT scans of the chest are useful in diagnosing acute thromboembolic disease.

- the ventilation-perfusion lung scan remains reliable in most centers for establishing the diagnosis of *chronic thromboembolic disease*.
- A high-resolution CT scan of the chest is the most accurate means of diagnosing emphysema and interstitial lung disease.

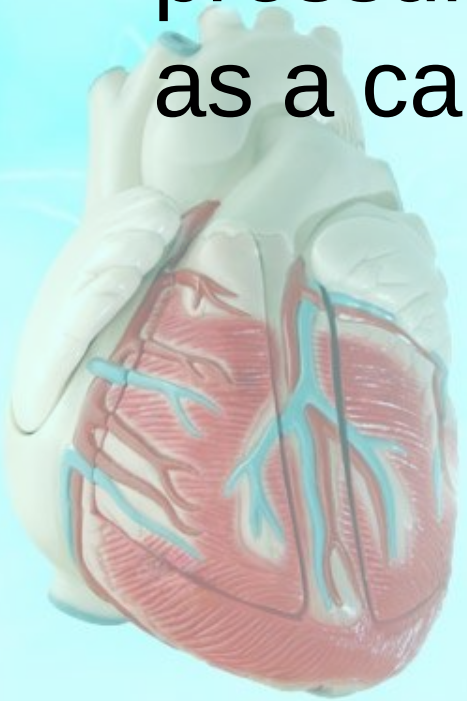
- Two-dimensional echocardiography is useful for measuring RV thickness and chamber dimensions as well as the anatomy of the pulmonary and tricuspid valves.
- The interventricular septum may move paradoxically during systole in the presence of pulmonary hypertension.



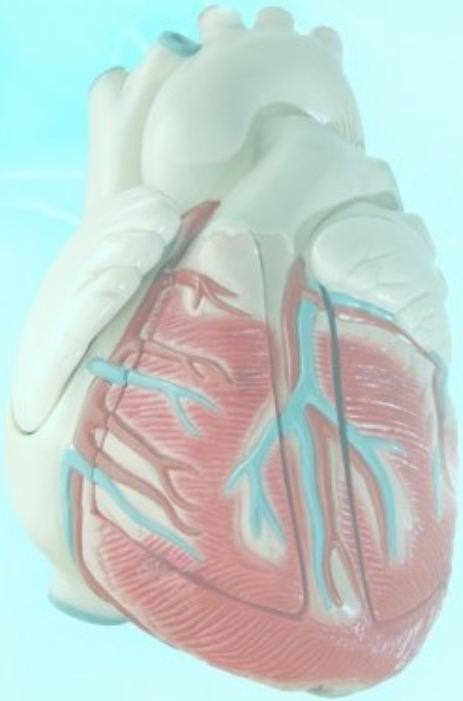
- Doppler echocardiography can be used to assess pulmonary artery pressures.
- MRI is also useful for assessing RV structure and function, particularly in patients who are difficult to image with 2-D echocardiography because of severe lung disease.



- Right-heart catheterization is useful for confirming the diagnosis of pulmonary hypertension and for excluding elevated left-heart pressures (measured as the PCWP) as a cause for right heart failure.



- BNP and N-terminal BNP levels are elevated in patients with cor pulmonale secondary to RV stretch and may be dramatically elevated in acute pulmonary embolism.

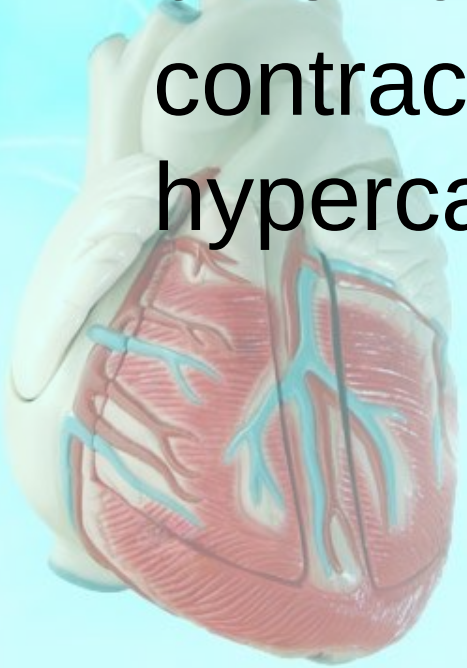


Treatment

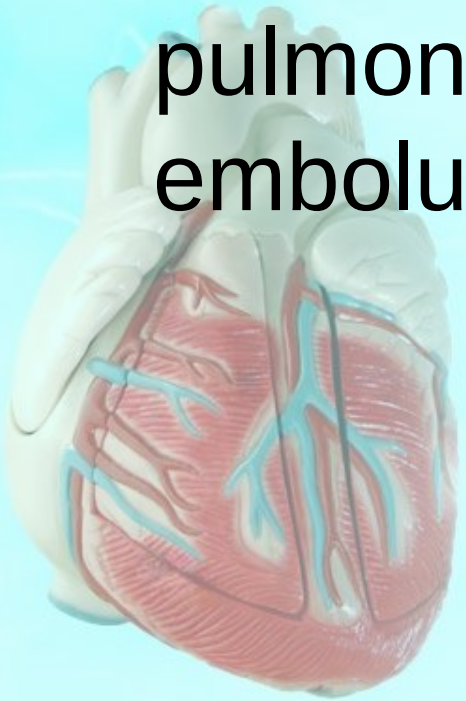
- The primary treatment goal is underlying pulmonary disease, since this will lead to a decrease in pulmonary vascular resistance and relieve the pressure overload on the RV.
- General principles of treatment include decreasing the work of breathing using noninvasive mechanical ventilation, bronchodilation, and steroids, as well as treating any underlying infection.

- Adequate oxygenation (oxygen saturation 90–92%) will also decrease pulmonary vascular resistance and reduce the demands on the RV.
- Patients should be transfused if they are anemic, and a phlebotomy should be performed to reduce pulmonary artery pressure if the hematocrit exceeds 65%.

- Diuretics are effective in the treatment of RV failure, and the indications for their use are similar to those for chronic HF.
- chronic diuretic use may lead to contraction alkalosis and worsening hypercapnea.



- Digoxin is of uncertain benefit in the treatment of cor pulmonale and may lead to arrhythmias in the setting of tissue hypoxia and acidosis.
- The treatment of the acute cor pulmonale that occurs with pulmonary embolus is described later.



reference

- **Harrison‘s**
  PRINCIPLES OF INTERNAL MEDICINE
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- Chapter 243 heart failure and cor pulmonale